IN THE SPECIFICATION

Please amend the specification by deleting the phrase "**BRIEF DESCRIPTION OF THE DRAWINGS**" on page 18.

Please amend the specification by deleting paragraphs 0044 to 0050 on pages 18-19.

Please amend the specification by replacing paragraph 0054 on page 21 with the following:

--A large Dutch family was used wherein hemochromatosis segregates as a dominant trait among family members ~~(Fig. 1)~~. The clinical symptoms in patients from this family were similar to complaints in other HH patients and include joint pains, osteoarthritis, fatigue, cardiomyopathies, endocrine disorders such as diabetes mellitus. For several markers on chromosome 2q, two point linkage analyses yielded positive lod score (Table 1).

Table 1

	Recombination fraction (θ)						
Marker	0	.01	.05	.10	.20	.30	.40
D2S335	0.77	1.36	1.75	1.74	1.40	0.89	0.32
D2S2314	1.90	1.87	1.72	1.54	1.14	0.71	0.27
D2S2273	0.69	1.13	1.50	1.51	1.22	0.78	0.29
D2S389		2.96	2.73	2.44	1.80	1.09	0.38
D2S2167	0.45	0.44	0.38	0.31	0.21	0.13	0.07
D2S117	1.60	1.64	1.70	1.62	1.27	0.78	0.27
D2S311	0.99	1.04	1.14	1.13	0.91	0.55	0.16
D2S2392	1.59	1.55	1.41	1.23	0.86	0.48	0.17
D2S2289	1.06	1.11	1.18	1.15	0.91	0.57	0.22
D2S325	0.90	0.90	0.94	0.95	0.81	0.52	0.19
D2S2382	1.12	1.09	1.00	0.87	0.61	0.36	0.13

Please amend the specification by replacing paragraph 0060 on page 24 with the following:

--The human SLC11A3 gene encompasses 20 kb and consists of 8 exons. The open reading frame of 1716 bp starts at position 305 of exon 1 and ends at position 314 of exon 8 ~~(Fig. 2)~~ encoding a protein of 571 amino acids with 9 or 10 transmembrane domains.--

Please amend the specification by replacing paragraph 0061 on pages 24-25 with the following:

--Although conflicting evidence exists in the prior art, it has been confirmed that human SLC11A3 is expressed in most tissues but especially in those tissues involved with iron metabolism. Accordingly, expression of SLC11A3 is highest in the digestive tract, liver, placenta, kidneys and monocytes ~~(Fig. 5)~~.--

Please amend the specification by replacing paragraph 0064 on pages 25-26 with the following:

--The mutation leads to an amino acid substitution of asparagine by histidine at position 144 (N144H). Linkage disequilibrium of this mutation with another yet unidentified mutation is possible but this is not likely for a number of reasons. First, no other sequence alteration segregating with the disease was detected. Second, due to the highly conserved nature of asparagine in vertebrates ~~(Fig. 4)~~, the substitution of

asparagine suggests a pronounced effect on SLC11A3 function.--

Please amend the specification by replacing paragraph 0065 on page 26 with the following:

--Several possible results arise from the mutation. Protein structure prediction programs predict that the mutation is expressed in a transmembrane domain which may explain the effect of the mutation on the SLC11A3 structure. Another result may arise from the fact that asparagine is a neutral amino acid, i.e. when asparagine is substituted with the polar histidine the hydrophobicity of the transmembrane domain may induce protein folding. Finally, it is clear that the mutation is important for metal ion binding because it resides within a region of the protein that contains other divalent metal transporters that otherwise show little homology to SLC11A3 ~~(Fig. 4)~~.--

Please amend the specification by deleting paragraph 0103 on pages 39-40.